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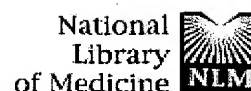
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Institute for Biomedical Aging Research of the Austrian Academy of Sciences, Rennweg 10, 6020, Innsbruck, Austria. Georg.Wick@oeaw.ac.at

Immune-inflammatory processes are increasingly discussed as possible pathogenetic factors involved in the development of atherosclerosis. Here, we summarize data on which we have built our "immunological" hypothesis of atherogenesis. This concept is based on the observation that nearly everybody shows protective cellular and humoral immune reactions against microbial heat shock protein 60 (HSP 60). Because a high degree of antigenic homolog exists between microbial (viral, bacterial, parasitic) and human HSP 60, this protective immunity may have to be "paid for" by the danger of cross-reactivity with human HSP 60 that is expressed by endothelial cells of stressed arteries. Arterial endothelial cells are more prone to produce HSP 60 and various adhesion molecules upon exposure to stress factors, including classical risk factors for atherosclerosis, due to their life-long exposure to the high arterial as compared to venous blood pressure. Also, endothelial cells are the first potential targets encountered by circulating HSP 60-specific T cells and antibodies. This concept not only opens new avenues for diagnostic approaches, but also may form the basis for new ways of therapeutic intervention.

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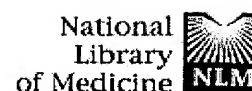
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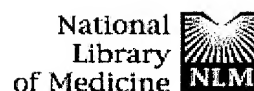


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

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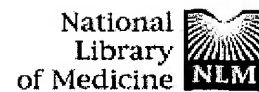
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